cytes in the lavage fluid and their numbers reliably reflect the intensity of sarcoid alveolitis. Because the activity of pulmonary sarcoidosis can be correctly judged by analyzing the bronchoalveolar fluid, this technique is extremely useful in staging sarcoidosis patients and monitoring their response to therapy.

Results of serum angiotensin-converting enzyme level, gallium 67 scan and bronchoalveolar lavage lymphocyte count not only correlate with clinical disease activity but also provide better understanding of biochemical and immunologic features of sarcoidosis. It has been suggested that the three markers probably reflect different stages of the granulomatous process. Further studies are needed to clearly define the indications for their routine use.

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The Clinical Use of Monoclonal Antibodies

IN 1975 Kohler and Milstein developed a relatively simple technique for producing large quantities of monoclonal antibody of desired specificity, that is, antibody secreted by a single cell and its clonal progeny, uncontaminated by other antibodies. One of the initial applications of this technology was the use of monoclonal antibodies to distinguish subpopulations of human T-lymphocytes that turn the immune response either on or off. In the peripheral blood of normal persons the proportion of T-cells expressing "helper" or "suppressor" markers is relatively fixed, possibly reflecting an appropriate balance of the functions of these subsets. In a number of disorders, such as multiple sclerosis and acquired immune deficiency syndrome (AIDS), these proportions are altered, and often the alterations correlate with disease activity. In recipients of organ allografts, the ratio of suppressor-to-helper cells may predict graft survival. In patients who have rheumatoid arthritis treated with total lymphoid irradiation, a dramatic increase in the ratio of suppressor-to-helper cells occurs with clinical improvement. Based on observations such as these, the monitoring of lymphocyte subsets with monoclonal antibodies will likely become an increasingly common practice.

The potential uses of monoclonal antibodies extend far beyond analyzing lymphoid cells. Monoclonal antibodies to human leukocyte antigen (HLA) markers such as HLA-B27 have already been produced and may prove useful in disease diagnosis and histocompatibility testing. It should be possible to produce monoclonal antibodies with specificity for any cell type, for use in histopathologic diagnosis or x-ray imaging. Antibodies to infectious agents or tumor-associated antigens can be used for immunodetection in vivo or in vitro.

Perhaps the most exciting of the potential applications of monoclonal antibodies is their use in vivo as therapeutic agents. Their appeal in this regard is due not only to their exquisite specificity for target antigens but also to the fact that antibodies can be attached to other compounds such as drugs, radioactive isotopes or toxins for delivery of those compounds to specific target tissues. Murine monoclonal antibodies to lymphocyte surface antigens have already been used in experimental protocols to treat leukemia and lymphoma, and the results have been encouraging. One limitation of mouse monoclonal antibodies is that they induce the formation of antimouse antibodies in human hosts, and these antiantibodies can neutralize the desired effect of a therapeutic antibody. Although this problem may be obviated by the use of human monoclonal antibodies, therapy with monoclonal antibodies must be limited to experimental settings until optimal protocols are developed. Nonetheless, there is little doubt that this new approach will ultimately contribute significantly to the treatment of a variety of disorders.

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Streptokinase and Acute Myocardial Infarction

EXTENSIVE MYOCARDIAL NECROSIS is a frequent cause of chronic heart failure and death in patients who have acute myocardial infarction. Measures designed to limit myocardial necrosis by limiting myocardial oxygen demand, increasing collateral blood flow or correcting various ischemic cellular defects have not proved satisfactory in the clinical setting. The need for restoration of antegrade flow in an occluded coronary artery has become obvious. Revascularization by emergency surgical bypass appears impractical; moreover, its ability to salvage a significant portion of jeopardized myocardium remains uncertain. Based on postmortem studies, in vivo angiography and intraoperative evidence that thrombosis is usually the cause of coronary occlusion in acute myocardial infarction, studies have been undertaken to determine if it is possible to reopen the occluded coronary artery rapidly by direct intracoronary administration of thrombolytic agents like streptokinase, urokinase or streptokinase plasminogen mixture for lysing occlusive clots. These studies have since confirmed that the arteries can—in 70 percent to 90 percent of cases—be reopened, usually within 30 minutes. Because the ruptured atherosclerotic plaque that initially triggered the thrombosis is still present, the artery must be protected by effective anticoagulation for about three months, initially with intravenous administration of heparin and, after five to seven days, warfarin. When the coronary artery was completely occluded and collaterals were not visible on angiography, significant myocardial salvage was usually not